JACC Vol. 5, No. 2
February 1985:366–8

CASE REPORTS

Mid-Diastolic Opening of the Pulmonary Valve After Right Ventricular Infarction

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Premature opening of the pulmonary valve in mid-diastole before atrial systole was observed during inspiration in a patient with extensive right ventricular infarction. Transient inspiratory elevation of right ventricular diastolic pressure to a level exceeding pulmonary artery pressure was documented. Although forward flow into the pulmonary artery was seen on Doppler echocardiography after atrial systole, most of the flow continued to be in response to right ventricular contraction, despite extensive infarction.

(J Am Coll Cardiol 1985;5:366–8)

Premature opening of the pulmonary valve in mid-diastole has been noted in a number of unusual conditions in which right ventricular pressure exceeds pulmonary artery diastolic pressure before atrial contraction. These cases have included sinus of Valsalva rupture into the right atrium (1), Uhl's anomaly (2), constrictive pericarditis, Loeffler's endocarditis, Ebstein's anomaly, pulmonary regurgitation and tricuspid valvulectomy (3). We report on a patient who demonstrated mid-diastolic opening of the pulmonary valve and late diastolic forward flow into the pulmonary artery after right ventricular infarction.

Case Report

A 64 year old man had an acute inferior wall myocardial infarction complicated by transient right bundle branch block, recurrent ventricular tachycardia, ventricular fibrillation and right heart failure with peripheral edema and jugular venous distension. He was thought to have had right ventricular as well as left ventricular infarction.

Cardiac catheterization showed elevation and equalization of both right and left heart diastolic pressures, with high grade obstructive lesions of all three coronary arteries.

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Coronary revascularization was performed with intraoperative mapping and attempted ablation of a focus of inducible ventricular tachycardia along with placement of two 25 cm^2 epicardial "patches" for possible later use with an automatic implantable defibrillator (AID-B, Intec Systems, Inc). The pericardium was not thickened or abnormal in appearance at the time of surgery.

Postoperatively, the patient continued to have exertional dyspnea with limited exercise capacity and required high doses of diuretic drugs to control peripheral edema.

Repeat catheterization. After surgery, cardiac catheterization demonstrated a cardiac index of 1.7 liters/min and increased (24 mm Hg) left ventricular diastolic, right ventricular, right atrial and pulmonary capillary wedge pressures with a "dip and plateau" pattern in the ventricular tracings and a prominent right atrial Y descent. Computed tomography showed normal pericardial thickness.

Echocardiography. M-mode, two-dimensional and Doppler echocardiography (Irex, system IIIB) showed a markedly dilated, diffusely hypokinetic right ventricle, dyskinesia of the entire interventricular septum and parts of the inferior wall of the left ventricle, no pericardial effusion or tricuspid regurgitation and intermittent mid-diastolic opening of the pulmonary valve.

Pulmonary valve. Pressure measurements could not be performed simultaneously with echographic examination of the pulmonary valve because of interference caused by the catheter lying across the valve. Figure 1 is a superimposition of pulmonary artery and right ventricular pressure tracings obtained by fluid-filled catheters moments before the pulmonary valve echogram was performed at an identical heart rate and at the same phase of respiration. At the onset of

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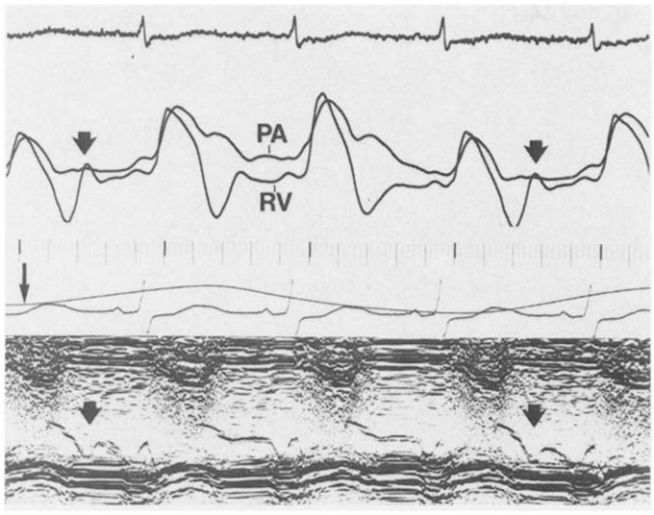

Figure 1. Pulmonary artery (PA) and right ventricular (RV) pressure tracings superimposed on the pulmonary valve echocardiogram recorded a few seconds later at an identical heart rate and at the same phase of respiration. With inspiration (**small wide arrows**), right ventricular mid-diastolic pressure transiently exceeds pulmonary artery pressure, causing the pulmonary valve to open (**large thin arrow**).

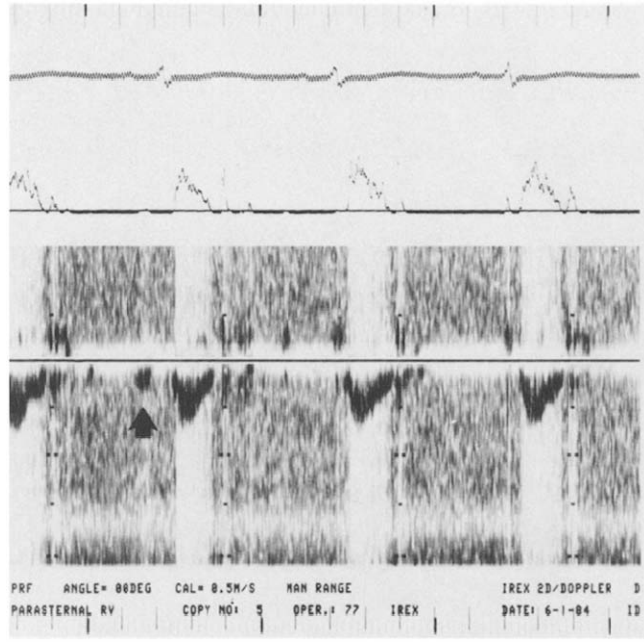

Figure 2. Doppler echocardiogram obtained from the left parasternal border with the beam directed into the main pulmonary artery. Flow away from the transducer into the pulmonary artery is displayed as a negative deflection. During inspiration, flow is seen after atrial systole (**arrow**). The majority of flow occurs after ventricular systole. CAL = calibration; PRF = pulse repetition frequency; RV = right ventricle; 2D = two-dimensional.

inspiration (Fig. 1, small arrow), right ventricular diastolic pressure transiently exceeded pulmonary artery pressure; this was associated with mid-diastolic opening of the pulmonary valve before atrial systole. During the remainder of the respiratory cycle, large A waves occurred after atrial systole, but mid-diastolic opening was absent.

A pulsed wave Doppler echocardiogram (Fig. 2) was performed from the left parasternal border with the sample volume in the main pulmonary artery. Intermittent inspiratory forward flow was present after atrial systole, but no mid-diastolic flow was detected.

Discussion

Our patient represents a case of pseudoconstrictive pericarditis caused by extensive right ventricular infarction. During inspiration, increased right heart filling caused an increase in right atrial and right ventricular pressures, with a simultaneous decrease in pulmonary artery pressure. The right ventricle was relatively noncompliant and nondistensible, not because of pericardial constriction, but because of infarction of the right ventricle with subsequent dilation and fibrosis. Whether the defibrillator patch electrode placed on the right ventricular free wall contributed to this process is speculative, but it is a possibility that cannot be excluded.

The Doppler echocardiogram suggested that little or no flow occurred through the open pulmonary valve in mid-diastole, but that with the additional force of atrial systole, forward flow into the pulmonary artery occurred before ventricular systole. Late diastolic forward flow occurred only during inspiration; however, the majority of pulmonary artery flow still occurred during right ventricular systole.

Previously reported cases. In their initial description of the clinical and hemodynamic features of right ventricular infarction, Cohn et al. (4) postulated that right atrial contraction might propel blood into the pulmonary artery during diastole or even that passive flow might occur in mid-diastole. Our patient demonstrated mid-diastolic opening of the pulmonary valve during inspiration, but no significant flow through the valve could be documented; forward flow did occur after atrial systole. However, the magnitude of inspiratory diastolic forward flow into the pulmonary artery was much less than systolic flow, indicating that right ventricular contraction, although seriously impaired, was still the primary means of propelling blood into the pulmonary artery.

Legrand and Rigo (5) reported on a patient with right ventricular infarction in whom premature opening of the pulmonary valve occurred only after atrial systole and probably varied with respiration. Coma-Canella et al. (6) reported on a case similar to ours, in which the pulmonary valve intermittently opened in mid-diastole before atrial systole in a patient with right ventricular infarction. They pos-

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tulated that this represented an inspiratory augmentation of right ventricular pressure to a level exceeding pulmonary artery pressure. Our case documents an inspiratory increase of right ventricular pressure above pulmonary artery pressure in mid-diastole with opening of the valve and forward flow occurring after atrial systole.

Role of echocardiography. The use of echocardiography in this case re-emphasizes the effectiveness of this technique in demonstrating cardiac structural abnormalities, providing indirect evidence of hemodynamic alterations and documenting functional changes in blood flow. Two-dimensional echocardiography confirmed right ventricular infarction with significant left ventricular involvement; M-mode echocardiography was used to precisely time the point of pulmonary valve opening, and Doppler echocardiography documented diastolic forward flow in the pulmonary artery. Echocardiography is thus a valuable aid in evaluating patients with myocardial infarction and can provide insight into the basic pathophysiologic mechanisms of right ventricular infarction.

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